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Acute Pancreatitis: Case Report and the Importance of Early Prediction of Severity

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INTRODUCTION

Acute Pancreatitis (AP) was the third most common GI diagnosis in 2012, resulting in approximately 275,000 admissions and costing about $2.6 billion.
It remains a disease characterized by significant morbidity and mortality, and to this day, there is not a medication to treat it. Most care is supportive.
Establishing the severity of the disease accurately is important in order to triage patients to the correct level of care to decrease rate of complications, mortality, and potentially shorten duration of stay[1].

CASE REVIEW

30yo male with history of alcohol use/abuse, with prior episode of pancreatitis 5mo earlier, presented to the Emergency Department with sudden onset of epigastric pain radiating to the back, nausea, vomiting. Patient's last drink was 4 days prior, admitting to binging over the weekend.

Initial VS: HR in 110s-120s, RR 20. BP 162/105.
Initial remarkable labs: WBC 18.3, Na 133, K 3.0, CO2 15, AG 25,BG 149 lipase 2610.

On physical exam, patient was uncomfortable with a diffusely tender abdomen. He appeared hypovolemic. He received 2L NS in ED.

Patient was admitted to the medical floor and started on 250mL/hr of LR, given Dilaudid for pain control and placed NPO.

8 hours after admit, patient acutely decompensated. Repeat **VS BP 92/60, HR 159, RR 25.** Abdomen was taught on exam. Patient was was transferred to the ICU for further management.

Repeat labs now showing **H&H 21&61, K 5.6, CO2 9, AG 21, Cr 1.35, CA 6.5, Mag 1.6, lactate 8.9**.

Patient was started on a bicarbonate drip, received calcium gluconate and 10 units of NPH, and D50 injection. A Foley catheter was inserted with an estimated bladder pressure of 12.

Despite measures, patient's acidosis and renal failure worsened and 3 hours later, patient was transferred to PPMC ICU for further management.

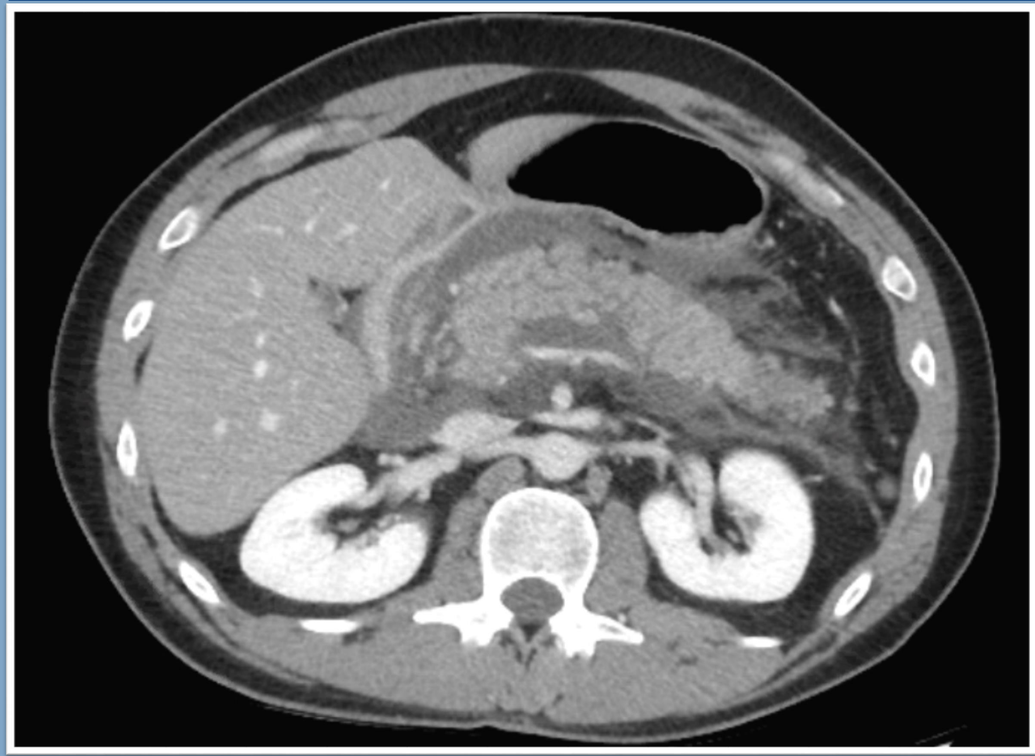
Revised Atlanta Classification of AP[4]

AP is divided into two broad categories:
- Interstitial edematous AP – no recognizable tissue necrosis
- Necrotizing AP – inflammation associated with pancreatic parenchymal necrosis and/or peripancreatic necrosis

According to severity, AP is divided as follows:
- **Mild AP:** absence of organ failure and local or systemic complications
- **Moderately severe AP:** transient organ failure (<48hrs) and/or local or systemic complications without persistent organ failure > 48hrs
- **Severe AP:** persistent organ failure that may involve one or multiple organs

Organ failure is defined as a score of 2 or more for any one of the 3 organ systems using the Marshal scoring system.

Imaging



Admission CT Abdomen and pelvis w contrast
Pancreas is enlarged and edematous.
There is extensive peripancreatic edema which causes significant narrowing of the upper most portion of SMV, portal vein, and splenic vein.
Fluid collections extend into the transverse mesial colon, anterior pararenal spaces, lesser sac, and porta hepatis.

Why predict the severity of pancreatitis?

The incidence of AP is increasing [1].
- 85% of patients with AP have acute interstitial edematous pancreatitis
- 15% of patient have necrotizing pancreatitis with necrosis of pancreatic parenchyma, the peripancreatic tissue or both.
- Overall mortality rate is 3% - 5% in patients with interstitial pancreatitis vs 17% in those with necrotizing pancreatitis

Predicting the severity of AP to triage patients to appropriate level of care is essential given most care is supportive. This is especially important in small hospitals that do not have an Intensive Care Unit.

DISCUSSION

Most authors of past and current guidelines recommend physicians to predict the severity of AP early on to guide the triage of patients.
A multitude of predictive models have been developed to predict the severity of AP based upon clinical, laboratory, and radiologic risk factors, various severity grading systems, and serum markers {2]
Some of these can be performed on admission to assist in triage of patients, while others can only be obtained after the first 48 to 72 hours or later.
However these predictive models have low specificity, which, when coupled with the low prevalence of severe AP, results in low positive predictive values.
- A CRP level above 150mg/L at 48 hours is associated with severe pancreatitis with an 80% sensitivity.
- A BUN level of 20mg/dL on admission is also associated with an increased risk of death, as is an increase in BUN at 24hrs.
- Ranson's criteria and Apache II scores are 2 scoring tools frequently used in inpatient medicine.
- Whilst an Apache II score has a median sensitivity of 100% and can be used on admission and repeated daily, a Ranson's score > 2 has a sensitivity and a specificity in the 80s, and has to be calculated at 48hours.
- An Apache II score > 8 and up-trending scores suggest a severe episode[5]
- Other scoring systems include SIRS, BISAP, CTSI.

Complications

Local complications:
- Acute peripancreatic fluid collection
- Pancreatic pseudocyst
- Acute Necrotic collection
- Walled-off Necrosis
- Portosplenomesenteric venous thrombosis in about 50% of patients with necrotizing AP.
- Pseudoaneurysm
- Abdominal Compartment Syndrome

Systemic complications:
- Systemic Inflammatory Response Syndrome
- Organ failure
- Death

KEY LEARNING POINTS

In terms of predicting severity of AP:
- Apache II score most sensitive in predicting severe AP[3]
- CRP and BUN can be use as adjuncts in determining severity of AP
- These scoring tools do not replace clinical judgment.

Future studies could study existing predictive markers and tools currently used in identifying moderately severe and severe AP in the initial 24-72hrs, and measure clinical outcomes in groups with and without the use of such tools.

Patient follow-up

The patient was transferred to PPMC 12hrs after ED presentation with rapid deterioration due to severe AP with third spacing/hemoconcentration (H&H 20&60), hypocalcemia, lactic acidosis.
Patient was intubated day 1 of hospitalization due to worsening acidemia, and developing moderate ARDS. He was also later that day started on CRRT with oliguria and worsening renal function.
Overnight his abdominal pressure reached 30mmHg then slowly decreased but never normalized.
Patient developed persistent hypotension despite 2 vasopressors.
Patient developed fulminant liver failure on day 4.
Patient passed away 5 days after presentation.

This case is particularly unique in that the patient experienced a very rapid deterioration and developed many of the rare complications associated with severe AP including portal vein thrombus, abdominal compartment syndrome, ARDS, and multiple organ failure leading to death.

Interestingly, his APACHE II score on admit to PMH ICU was 12 points, thus predicting a mortality of 15%.
Repeated at 48hrs, his APACHE II score was 27 points (55% predicted mortality), whilst his Ranson's score was 5, a 40% predicted mortality. In our case, the Apache II score was indeed more sensitive in predicting the associated mortality.

References

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